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UNILATERAL PARALYSIS AGITANS OCCURRING AFTER HEMIPLEGIA.

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Unilateral paralysis agitans, so-called, is not an uncommon condition. Two distinct forms can be recognized: that in which the more obvious symptoms of the disease appear first on one side, or are at least more pronounced upon one side, and that in which the paralysis agitans has developed equally, but subsequently has, in consequence of hemiplegia, ceased, either temporarily or permanently, upon the paralyzed side. I have been unable to discover the record of any case in which, some years after an attack of hemiplegia, paralysis agitans has developed and remained restricted to the healthy side. The following instance of this condition, therefore, seems worth reporting.

Mrs. D., now 76 years of age, had, at the age of 69, a stroke of apoplexy, causing paralysis of the right side. Speech was affected; the mouth was drawn to the right, and the leg and arm were paralyzed, but apparently incompletely. The speech soon became normal again, and there was a considerable return of power in the right arm and leg. At the age of 73—four years after her attack—she began to notice shaking in the left arm and hand, which has continued to the present time, apparently increasing very moderately in severity.

Owing to the somewhat imperfect memory of the patient, the family history is untrustworthy, and the early previous history, possibly for the same reason, appears to be unimportant. The present condition is as follows:

She is very stout; the legs are edematous; the peripheral arteries are in a state of advanced sclerosis; there is a loud systolic murmur, heard best at the aortic cartilage, and transmitted clearly into the vessels of the neck. Speech is apparently normal, although the patient claims that she cannot speak as well as she did a year or two ago. The pupils are equal, and react to light, but she states that her eyesight has failed recently. The expression is mask-like. In the left hand there is a persistent tremor. At the wrist this consists in movements of extension and flexion; the thumb is in a position of apposition, and, with the fingers, exhibits the characteristic pillroller's tremor. When the patient attempts a voluntary movement the tremor stops completely, and may remain absent for as long as two minutes, but if the movement continues for a longer time the tremor reappears, at first faintly, but soon becoming more violent than during rest. There is no tremor whatever of the right arm or hand during either rest or movement. There is moderate impairment of power in both arms and hands, apparently about equal in degree, but the movements of the right arm appear to be more limited than those of the left. The patient states that within a year the left arm was considerably stronger than the right. There is a tremor in both feet at the ankle, resembling the oscillations of clonus. This tremor disappears if the feet are forcibly flexed dorsally. The tendon reflexes of the arms and legs are absent. Distracting the patient's attention by asking her to count or look at the ceiling does not cause them to appear, and Jendrassik's method cannot be tried. The Babinski reflex is not present on either side. Walking is impossible, probably in part due to weakness, to the severe edema of both legs, and to the excessive weight. While sitting in her chair there is no sign of either propulsion or retropulsion. There is distinct rigidity to passive movement of the left arm.

To summarize the case briefly: Seven years ago a woman of 69 developed right hemiplegia, from which she made a partial recovery. Four years later she first noticed tremor in the arm and hand of the opposite side, which has continued until the present time. There is now the typical symptom-complex of paralysis agitans in the left arm, with paresis of the left arm, and tremors in both feet. In short, there is an incomplete Parkinsonian hemiplegia, and some traces of an old organic hemiplegia.

The first observation of a case of unilateral paralysis agitans is credited to Marshall Hall, but as the tremor in his case (a young man) developed only upon voluntary motion, then becoming violent, and as there were also curious movements of the eyes, and disturbances of speech, it is fair, I think, to conclude that his patient was suffering from multiple sclerosis. Later observers, however, have generally united in believing that paralysis agitans, at least in its incipiency, is nearly always unilateral, although they all state that in the course of a few years it becomes generalized. (Brissaud, Eulenberg, Erb, Berbez, Wollenberg, Williamson, and Gowers.) Fuerstner is the only author who states positively that the disease may be limited to one side throughout its entire course. Dutil records a case of 2 years' duration, atypical in some respects, in which the tremor had remained limited to the left side. The other symptoms were general in character. Berbez, in 28 cases examined in Charcot's clinic, found 3 cases in which the tremor was unilateral, and 7 in which it was more pronounced on one side than on the other. The three unilateral cases were in the early stage, and there was no reason to believe that extension might not ultimately take place.

Regarding the arrest of the tremor by hemiplegia, there seems to be very little definite information in the literature. Eulenberg and Lamy, both of whom have written comprehensive articles on the subject for systemic works, have failed to mention it at all. On the other hand, Gowers, Wollenberg, and Williamson speak of it as a well-known fact. Gowers states that, if the paralysis is not complete and permanent, the tremor may return, and Williamson states that the arrest of the tremor after cerebral hemiplegia is usually transient. Williamson makes the assertion that there are two cases on record in which the arrest of the tremor was permanent. Examination of the literature cited in the monographs of Wollenberg and Williamson has failed to show upon which actually recorded cases their statements were based.

The practical value of observations of unilateral Parkinson's disease, and the effects of hemiplegia upon the tremor, consists of its aid in the determination of the localization of the lesion. This localization has been made in every part of the motor tract, from the cerebral cortex to the muscular fibers.

The results of histological examination and investigations have been contradictory. Changes have been found in the spinal cord similar to those found in the cords of old persons suffering from general arteriosclerosis, but these changes are by no means constant, observations by competent neuro-pathologists having failed to reveal any changes at all. (Fuerstner.) No characteristic changes whatever have hitherto been found in the cerebral cortex, although the majority of neurologists now believe that the lesion is situated there. A few, basing their opinion upon some of the symptoms, and the fact that occasionally tumors of the peduncle have caused symptoms resembling paralysis agitans, believe that it is in the basal ganglia. Blocq and Marinesco believe that the lesion affects chiefly the motor tract in the lower portions of the brain.

It seems hardly worth while to repeat the various arguments in favor of the cerebral localization. That based upon the unilateral occurrence of the disease is not entirely satisfactory because, in disseminated sclerosis, even when the cord is chiefly involved, the symptoms may be preponderant upon one side, and the fact that paralysis agitans almost invariably invades the other side, would be rather in favor of a general brain disease, or at least a disease affecting the brain bilaterally, even if unequally, than a disease affecting one hemisphere. It is easy to understand how diffuse lesions of the pons or medulla might, under exceptional circumstances, produce tremor only upon one side of the body. In practically all recorded cases there have always been symptoms that show that the opposite side was sharing in the disease. Neither does it seem to me that the cessation of the tremor, after hemiplegia, necessarily indicates a cortical situation. It merely suggests strongly that the cause of the tremor affects the central motor neurones, not the peripheral neurones. And this view is supported by the frequent reappearance of the tremor with the restoration of power, which means a recovery of the central motor neurones. It is of somewhat more significance that the moderate injury of these central neurones may serve to check the subsequent development of the tremor in the injured side.

There is no question that, in a part that has been subject to a tremor, it can be more readily produced than in a part in which tremor has never occurred. In the same way, the tremor of paralysis agitans, if inhibited for a time by a sudden injury to the central motor mechanism, may be apparently easily re-established; but if the central motor mechanism has never learned to produce a tremor, then it is unable to acquire it after a moderate degree of injury.

Of course, in the present case, although the time that has elapsed since the onset of the tremor in the left arm is rather longer than usually occurs without the development of a tremor in the right arm, no definite statements can be made as yet. It is still possible that bilateral tremor will occur, just as, seemingly, it has already occurred in the feet; but even if it should subsequently occur, it will be justifiable to ascribe the long delay to the hemiplegia.

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